REMARKS/ARGUMENTS

Claims 4, 7-10, and 25 are pending in this application.

Support for the amendment to Claim 9 is found in the paragraph bridging pages 2-3 in the specification.

Claim 26 has been amended to clarify that decreasing or increasing vasopressin level is what is done in the method claimed (see, e.g., page 2, 4th paragraph in the specification).

No new matter is believed to have been added by this amendment.

The objections are no longer applicable as Claims 16 and 19 are cancelled, Claim 25 has been amended as suggested, and Claim 26 has been amended to provide decreasing or increasing--maintaining has been removed.

Withdrawal of those objections is requested.

The rejection under 112, first paragraph pertaining to enablement is respectfully traversed. That is, Claim 9 defines treating at least one of four specific symptoms-- polyuria, dehydration, mouth dryness and hyperosmolarity, by administering an antibody or binding fragment thereof specifically binding to SEQ ID NO:75. The specification does describe general methods of making antibodies, something which is well-known in the art, and a specific antibody-producing hybridoma (see Claim 6). To treat those symptoms then, all one has to do is administer those antibodies with specifics such as dosages, regimens, etc being determined by the clinician as is customarily the practice based on age, weight, race, gender, and other physiological factors of the patient. With respect to Claim 26, again the operative step is to administer the antibody that binds to SEQ ID NO: 75, the resulting decrease or increase in vaspopressin will follow as is described in the specification.

The rejection under 112, second paragraph is respectfully traversed. Claims 9 and 26 have been amended to provide the increase or decrease of blood vaspopressin. Claim 16 has been cancelled.

To the art-based rejections citing US '194 or CA '332, by themselves or combined with Kitamura or Harlow and US '778 and to the obviousness-type double patenting rejections in view of US '194 by itself or with Kitamura, Harlow and/or US '778.

The fundamental basis for these rejections is that of alleged inherency. Again, the Examiner has noted that art "inherently" achieve the effects on vasopressin levels. However, the Examiner has provided no proof of this. Rather, the Examiner is using Applicants' disclosure against them. As noted by the court in *In re Oelrich*, 666 F.2d 578, 581, 212 USPQ 323 (CCPA 1981), the mere fact that a certain thing may result from a given set of circumstances is not sufficient to prove inherency. Inherency may not be established by probabilities or possibilities. Something that is inherent must inevitably be the result each and every time.

It is by now well settled that the burden of establishing a *prima facie* case of anticipation resides with the Patent and Trademark Office. *In re Piasecki*, 745 F.2d 1468, 1472, 223 USPQ 785, 788 (Fed. Cir. 1984), quoting *In re Warner*, 379 F.2d 1011, 1016, 154 USPQ 173, 177 (CCPA 1967).

As noted by the Board of Patent Appeals and Interferences in *Ex parte Skinner*, 2 USPQ2d 1788, before an Examiner can switch the burden of proof of showing non-inherency to the applicant, the Examiner must provide some evidence or scientific reasoning to establish the reasonableness of the Examiner's belief that the functional limitation is an inherent characteristic of the prior art. In this case, the Examiner has provided no such evidence.

It is Applicants' position that the treatment of hypercalcemia bears no immediate relationship to vasopressin levels in blood. That is, the present application is the first to show that PTHrP causes a decreas in vasopressin level in blood, and that administration of anti-PTHrP antibody increase in vasopressin levels in blood. Applicants are to provide experimental results demonstrating that anti-PTHrP antibody when administration resulted in an increase in vasopressin. However, if administered Alendronte (sodium [4-amino-1-hydroxy-1-(hydroxy-oxido-phosphoryl)- butyl]phosphonic acid trihydrate), this did not effect vasopressin levels in the blood. Alendronate is a typical therapeutic agent for treating humoral hypercalcemia of malignancy (HHM) thus indicting that therapeutic effects on HHM bears no immediate relationship to vasopressin in the blood.

Further, the art does not provide any insight into treating patients having at least one symptom selected from the group consisting of polyuria, dehydration, mouth dryness and hyperosmolarity (see Claim 9 and Claim 29).

Reconsideration of the outstanding rejections in light of the amendments and remarks contained herein is requested.

Respectfully submitted,

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